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Epidemiology of Health Effects of Radiofrequency Exposure

ICNIRP (International Commission for Non-Ionizing Radiation Protection) Standing Committee on Epidemiology:

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Abbreviations:

APC	Adaptive power control
CI	Confidence interval
DNA	Deoxyribonucleic acid
FM	Frequency modulation
Hz	Herz
Km	Kilometer
OR	Odds ratio
RR	Relative risk

SAR	Specific absorption rate
SIR	Standardized incidence ratio
SMR	Standardized mortality ratio
TV	Television
US	United States of America
W	Watt

Abstract

We have undertaken a comprehensive review of epidemiological studies about the effects of radiofrequency fields (RF) on human health in order to summarize the current state of knowledge, explain the methodological issues that are involved, and aid in the planning of future studies. There have been a large number of occupational studies over several decades particularly on cancer, cardiovascular disease, adverse reproductive outcome, and cataract in relation to RF exposure. More recently there have been studies of residential exposure mainly from radio and TV transmitters, and especially focusing on leukaemia. There have also been studies of mobile phone users particularly on brain tumours and less often on other cancers and on symptoms. Results of these studies to date give no consistent or convincing evidence of a causal relation between RF exposure and any adverse health effect. On the other hand, the studies have too many deficiencies to rule out an association. A key concern across all studies is the quality of assessment of RF exposure. Despite the ubiquity of new technologies using RF, little is known about population exposure from RF sources and even less about the relative importance of different sources. Other cautions are that mobile phone studies to date have only been able to address relatively short lag periods, almost no data are available on the consequences of childhood exposure, and published data largely concentrate on a small number of outcomes especially brain tumour and leukaemia.

Introduction

The advent of mobile phones, now used by about 1.6 billion people worldwide, has been accompanied by an upsurge in public and media concern about the possible hazards of this new technology, and specifically of radiofrequency field (RF) exposure. Although some epidemiological research was conducted several decades ago on RF in occupational settings, in general the effects of RF in humans are an emerging area of investigation, and most studies are recent or not yet published. Furthermore, although the results of studies of mobile phone risks have received widespread public attention, their interpretation is not straightforward because of methodological difficulties. In particular, because RF is invisible and imperceptible individuals cannot directly report on their exposure, and therefore the quality of exposure assessment needs particularly careful consideration when interpreting epidemiological studies. In order to summarize the current state of knowledge, to explain the methodological issues that need to be considered when assessing studies, and to aid in planning future studies, we have undertaken a broad review of epidemiological knowledge about the effects of RF on human health. We have divided the literature, for this purpose, into studies of RF exposure from occupational sources, from transmitters, and from mobile phones.

This review covers the possible effects of long-term exposure to RF - defined as 100 KHz to 300 GHz - on the risk of diseases: for instance, cancer, heart disease and adverse outcomes of pregnancy. We have not reviewed the health consequences of communications technology that are indirect or unlikely to be due to radiation. In particular, RF can interfere with implanted medical devices, such as cardiac pacemakers, but the effects on health are a consequence of this interference, rather than a direct effect on the body; phone conversations by drivers of moving vehicles appear to raise the risk of motor vehicle accidents, but this is

probably related to distraction rather than RF exposure. While anxieties and psychosomatic illnesses might be caused by knowledge of the presence of phones or phone masts, again this would not be an effect of RF and is not discussed.

As well as epidemiological studies of disease causation some studies have been published that use an epidemiological design to investigate whether mobile phones can affect acute symptoms, such as headaches. For completeness we have included these in this review, although such investigations are usually better conducted by laboratory volunteer experiments rather than by observational epidemiology, given the high degree of susceptibility to biased reporting in response to concerns.

Because this is primarily an epidemiological review we have not detailed the physics and dosimetry of RF from different sources, which are described elsewhere (Hitchcock and Patterson 1995; IEGMP 2000; Mantiply et al. 1997) However, because understanding of mobile phone-related epidemiology is critically dependent on understanding of mobile phone technology, we have included some information explaining this technology. We have also included, because of its importance to future research advance, some comments on the interface between physics and epidemiology, and the gaps to be bridged between these disciplines if more rigorous investigation of potential RF effects is to be achieved.

Exposure

Sources of exposure

Communications sources have increased greatly in recent years, and there is continuing change in the frequencies used and variety of applications. The first mobile phone systems

were analogue and utilized 450 and 900 MHz. Digital systems, operating at somewhat higher frequencies (1800-1900 MHz) and using different modulation techniques, became prevalent in the early 1990s. Currently, the third generation systems (3G) using the Universal Mobile Telecommunication System (UMTS) are being introduced, which will operate in the 1900-2200 MHz frequency range. Occupational RF exposures occur to workers engaged in a number of industrial processes, particularly when using dielectric heaters for wood lamination and the sealing of plastics and industrial induction heaters. Relatively high levels of exposure to RF fields can occur to workers in the broadcasting, transport and communications industries, and the military, when they work in close proximity to RF transmitting antennas and radar systems. Medical exposures can come from medical diathermy equipment to treat pain and inflammation, electrosurgical devices for cutting tissues, and diagnostic equipment such as Magnetic Resonance Imaging (MRI).

Distribution of exposure in the population

Despite the rapid growth of new technologies using RF, little is known about population exposure from these and other RF sources and even less about the relative importance of different sources. In a typical house, non-occupational exposure could come from external sources, such as radio, TV, and mobile phone base stations, as well as internal sources, such as a faulty microwave oven, in-house bases for cordless phones, or use of mobile phones.

Radio and TV transmitters have a large coverage area and therefore operate at relatively high power levels up to about 1MW (Dahme 1999). Although these transmitters could generate fairly high fields at ground level, most are not located in heavily populated areas and do not lead to high exposure of the population.

Mobile phone base stations are low-powered radio transmitters that communicate with users' handsets. In early 2000, there were about 20,000 base stations in the United Kingdom and about 82,000 cell sites in the United States. Base stations can transmit power levels of 100 W or more (Schüz and Mann 2000). It is expected that the number of base stations will roughly double to accommodate new technology and a larger percentage of sites will have to be shared between operators, complicating exposure assessment. The power density levels inside a building can be from 1 to 100 times lower than outside, depending on the type of building construction (Schüz and Mann 2000). In addition, exposure can vary substantially within the building. For example, exposure was found to be about twice as high (and more variable) in the upper compared with the lower floors of a building (Anglesio et al. 2001). Driven by a typical pattern of use, the exposure from base stations shows a distinct diurnal pattern, characterised by lowest values during the night and by two maxima during the day, the first from 10 a.m. to 1 p.m. and the second from 6 to 10 p.m. (Silvi et al. 2001). There have been few and limited efforts to characterise population exposures; all of them have been small (usually areas around 10-20 base stations) (Anglesio et al. 2001; COST244 bis 2001; Schüz and Mann 2000). The total power density from the base stations was slightly higher than, but comparable with, the background power density from all other RF sources combined.

Mobile phones operate at a typical power of 0.25W. Analogue systems operated at higher power levels than the newer digital systems. Similarly older cordless phones operated to the analogue standard, while modern ones operate to the digital with a transmitted power of a base around 0.09W in a home but higher in a business setting. The actual exposure of the user depends on a number of factors such as characteristics of the phone, particularly the type and location of the antenna; on the way the phone is handled; and most importantly, on the

adaptive power control (APC), which may reduce the emitted power by orders of magnitude (up to a factor of 1,000). Factors that influence APC include distance from the base station, the frequency of handovers and RF traffic conditions. Thus the emitted power is higher in rural than in urban areas and when the user is moving (e.g. in a car). In areas where there is a great deal of phone use, phones may operate more than half of the time at the highest power levels. To compensate for the shielding effect of materials, power levels of phones are, on average, higher when a phone is used indoors than outdoors. RF absorption is maximal on the side of the head to which the phone is held, greatest close to the antenna, and falls off to less than a tenth on the opposite side of the head (Dimbylow and Mann 1999).

In an occupational setting, higher exposures occur, albeit infrequently; for example, radar exposed workers in the US Navy had potential for exposures greater than 100 mW/cm^2 (Groves et al. 2002).

Epidemiological considerations in exposure assessment

General: In the absence of information on what biological mechanism is relevant, it is unclear what aspect of exposure needs to be captured in epidemiologic studies. Because heating is the only known effect of RF, most research has assumed that the metric of choice must be a function of the Specific Absorption Rate (SAR). Metrics used in epidemiologic studies of other agents, such as cumulative exposure, average exposure over specific time intervals, and peak exposure need to be considered. Given the uncertainty about the relevant interaction mechanism, the dose needs to be assessed not just as external field intensity, but also as SAR for specific anatomical sites. Integrating exposure over time is further

complicated by the fact that sources vary markedly over very brief time periods relative to the time periods of interest.

Epidemiologic studies thus far have relied on rather crude proxies for exposure, such as job title, proximity to a base station, or use of a mobile telephone. Refinement of exposure assessment is critical to improved epidemiology. This requires a bridge between the rather disparate worlds of epidemiology and physics. While it is of interest to know about sources of variation or uncertainty in general, the critical need in epidemiological studies is to identify those variables that are most important in determining exposure levels and most amenable to capture within populations.

A key element in linking the complexity of the exposure sources and patterns with the needs of epidemiology is a meter that is capable of monitoring individual exposure. Such meters have now been developed (NRPB 2003).

Ideally, the dose, time pattern, and frequencies (wavelengths) of exposure from all key sources should be estimated for each individual in the study. Dose- and duration-response analyses are important to assessment of aetiology, but have often been absent in the existing literature (Swerdlow 1999). In addition, the possible lag period between exposure and disease manifestation needs to be considered. Hand-held mobile phones were not used regularly until the 1990s. Thus, studies published to date have had little power to detect possible effects involving long induction periods, or effects from long-term heavy exposure to mobile phones or base stations.

Methodologically, it would be desirable to conduct studies to clarify the relative contributions of different spheres of life. Such knowledge would allow epidemiologists to design studies that incorporate all important sources of RF exposure, or at least determine how much it matters that the occupational studies to date have taken no account of residential or mobile phone exposures and vice versa.

Occupational exposures: Most occupational epidemiological studies have based their exposure assessments simply on job titles and have included no measurements (see Tables 1, 2, 3, 4). It is possible that some jobs, e.g., radar operator, are adequate indicators of RF exposure. However, many job titles that have been previously considered to indicate exposure may provide a poor proxy for RF exposure.

In addition to improving exposure assessment in individual studies, there is the potential to develop job-exposure matrices, with the rows corresponding to relatively homogeneous groups with respect to RF exposure, defined by job title, perhaps specific work location, calendar time, and other recordable work history, and the columns corresponding to RF exposure metrics.

Transmitter exposures: All published epidemiological studies of transmitter exposures have based exposure assessment solely on distance from the transmitter. The relation between exposure and distance from the antenna is usually very complex, especially in urban areas. Close to the antenna, the field is very low due to the directional antenna characteristics. As one moves away, the field pattern can be complicated, with peaks and valleys in field intensity with increasing distance from the antenna. Röösli et al. (Röösli et al.) found no correlation between measured field values at random locations and distance from the base

station. This suggests that distance is a poor predictor of exposure and thus is of no, or little, use.

Estimation of community exposure to RF from transmitters may, however, be amenable to refinement. Geographic information systems allow for precise assignment of residence, topography, and some other likely determinants of exposure. Historical information on power output from the transmitters may well be available. This information combined with personal measurements may provide refined measures of exposure that can be applied retrospectively, with empirical validation.

Mobile phones exposures: Studies on mobile phones have used the simple dichotomy of user versus non-user, with some incorporating information on years of use, number of phone calls per day and duration of calls. Some studies have separated analogue and digital phone use. Few have included use of cordless phones, which also generate RF fields but from which exposure pattern is different and exposure generally much lower.

Ongoing studies are attempting to incorporate information on intensity of use, place of use, position of the telephone, type of telephone, and calendar period of use. Each of these extensions need to be evaluated, however, to determine (a) whether they are truly an important determinant of exposure and (b) whether they are amenable to accurate historical reconstruction through recall or some type of written record There is little benefit in knowing that the intensity of exposure varies by a parameter that cannot be captured, or gathering relatively precise information about, say, model of mobile phone, if no useful exposure variable can be derived from it.

Mechanisms

Heating of cells and tissues from RF exposure might have benign or adverse biological effects. These effects, which reflect an imbalance in the amount of heat built up in the body and the effectiveness of mechanisms to remove it, can be due to either elevated temperatures or increased physiological strain from attempts to remove the heat. Of particular concern for whole body heating are effects in the elderly, people taking certain kinds of drugs, and the foetus and infant. Cardiovascular mortality, birth defects and impaired ability to perform complex tasks are among the outcomes that have been associated with whole body heating. The sensitivity of different tissues and cells to thermal damage from both localized and whole body heating varies. The central nervous system, testis and lens of the eye seem to be particularly sensitive, the last due to a limited capacity to dissipate heat rather than due to a greater sensitivity of its cells to heat-induced damage.

Laboratory studies suggest that adverse biological effects can be caused by temperature rises in tissue that exceed 1°C above their normal temperatures (Goldstein et al. 2003). In addition to the absolute increase in temperature, duration of heating and thermoregulatory capacity of the body are important determinants of the harmful levels of tissue heating. High rates of physical activity, and warm and humid environments, will reduce tolerance to the additional heat loads.

There has been concern about possible carcinogenic effects of RF below levels that cause detectably harmful heating. RF is not sufficiently energetic to destabilize electron configurations within DNA molecules. Thus, there is no direct link between RF exposure and

genotoxic effects such as DNA mutations, DNA strand breaks, or other genetic lesions. Experimental evidence from animal and laboratory studies at the cellular level confirm the lack of genotoxic effect of RF (Krewski et al. 2001; Moulder et al. 1999). Similarly, an investigation in rodents did not find support to the suggestion that growth of tumours induced by other agents may be promoted by RF from mobile phone signals (Imaida et al. 2001; Mason et al. 2001).

Repacholi et al. (Repacholi et al. 1997) evaluated the effects of radiofrequency fields on tumorigenesis in a moderately lymphoma-prone Eμ-*Pim1* oncogene-transgenic mouse line. Exposure was associated with a statistically significant 2.4-fold increase in the risk of developing lymphoma. Utteridge et al. (Utteridge et al. 2002) recently repeated this study with a larger number of mice and with several refinements in the experimental design and did not demonstrate any difference in the incidence or type of lymphomas that developed between control and treated groups. Questions have been raised about the conduct and reporting of both studies and the inconsistency has not been resolved (Goldstein et al. 2003). Additionally, extrapolating the transgenic model to humans remains controversial.

Outcomes

A particular public concern appears to be that the use of hand-held mobile phones may be linked to the occurrence of malignant disease, especially brain cancer and, to a lesser extent leukaemia. Other tumours such as acoustic neuroma that occur in the head and neck region have also been investigated. Each of these conditions is rare. The incidence of malignant tumours of the brain in the general population is around 10 to 15 per 100,000 each year (Behin et al. 2003), the annual incidence of benign extra-cerebral tumours such as meningiomas is about 3 per 100,000, and benign tumours of the cranial nerves such as

acoustic neuromas, are rarer still. Because tumour incidence is so low, investigators have so far relied on case-control studies or, in a few instances, retrospective cohort studies. In addition, different tumour subtypes are likely to have different causes, as evidenced among brain tumours by the different molecular pathways leading to malignant astrocytomas on the one hand and benign meningiomas and acoustic neuromas on the other (Inskip et al. 1995). Similarly there are a variety of types of leukaemia each probably with differences in causation, making it even more difficult to ascertain sufficient numbers of homogeneous tumours for study. Epidemiological assessments have been further complicated because the environmental risk factors for malignant and benign brain tumours (Inskip et al. 1995), and hence potential confounders, are largely unknown beyond high-dose ionizing radiation. For leukaemia (Petridou and Trichopoulos 2002) knowledge of potential confounders is greater but still limited. Other risk factors, besides ionizing radiation, include exposure to chemotherapy, cigarette smoking, and benzene, as well as constitutional chromosomal abnormalities among children in particular.

Available evidence suggests that induction of brain tumours occurs over decades following tumorigenic exposures early in life. Latency of tumours varies from months to years depending on how aggressive tumour growth is and the location of the tumour. Epidemiological studies should therefore in principle allow for a lead time between potentially causal exposure and disease, although in the absence of biological or epidemiological evidence it is unclear what length this should be for potential RF effects.

Other chronic diseases such as cardiovascular disease, as well as symptoms, both acute and chronic, have been studied in relation to RF exposure. Headaches and other cranial discomforts including sensations of local warmth or heating, dizziness, visual disturbances,

fatigue and sleeplessness are the main symptoms reportedy users of mobile phones. All of these are common symptoms in humans.

Review of studies on occupational exposure

Cancer

Information on cancer risks in relation to occupational RF exposure comes largely from three types of epidemiological study: cohort studies, investigating a wide range of cancer (and non-cancer) outcomes in groups with potential RF exposure (Tables 1 and 2); case-control studies of specific cancer sites, investigating occupational RF as well as other exposures (Table 3); and analyses of routinely collected datasets on cancer incidence or mortality, in which risks of cancer have been assessed in relation to job title (Table 4). The most extensive literature addresses brain tumours and leukaemia.

Considering study size, design, and likely quality of RF assessment, the most informative studies (Groves et al. 2002; Milham 1988; Morgan et al. 2000) provide little evidence of an association with either brain tumours or leukaemia. The one possible exception was a raised risk of non-lymphocytic leukaemia in radar-exposed navy veterans (Groves et al. 2002) restricted to only one of three highly exposed occupations (aviation electronics technicians), but this finding was divergent from that of an earlier study of US naval personnel (Garland et al. 1990). Two US case-control studies of brain tumour aetiology have shown elevated odds ratios of around 1.5 in relation to jobs believed to have RF exposure. However, the study by Thomas et al. (Thomas et al. 1987) was based on interviews with relatives of dead cases, and hence was unable to identify exposure with much certainty. The other study (Grayson 1996) assessed exposures by a job exposure matrix based on historical reports of incidents of

exposure above permissible limits (10 mW/cm^2). No clear or consistent trend was found in risk of brain tumour in relation to exposure score. A widely cited study of US Moscow embassy staff and their dependents with possible RF exposure was only published as a précis by a third party (Goldsmith 1995); this leaves the study methods unclear, but few brain tumours or leukaemia occurred, and half were in dependents who lived outside the embassy.

A key concern across all these studies is the quality of assessment of RF exposure, including the question of whether it was truly present at all, and if so, for what proportion of the cohort. Although the published studies do not give consistent evidence for a raised leukaemia or brain cancer risk, they cannot be counted as substantial evidence against a possible association. Most of the studies suffer from severe imprecision, with the cancers of greatest interest rarely found in cohort studies of modest size and the exposure of interest rarely found in geographically based case-control studies. The cohort studies generally lack data on other relevant exposures, including non-RF frequencies of radiation, as well as on RF exposures outside the workplace (e.g., mobile phones). The studies based on routine data are vulnerable to publication bias given the many datasets worldwide that could be used to address this issue. Several of these studies did not follow workers after they left the job of interest (Garland et al. 1990; Grayson 1996; Szmigielski 1996), with the potential for bias if individuals left employment because of health problems that subsequently turned out to be due to cancer – this might especially be a problem for some types of brain tumour, which can be present for long periods before diagnosis. In addition, several studies have had substantial methodological inadequacies – for instance one study that found apparently raised risks for many different cancers, used more sources of exposure information for cancer cases than for non-cancer subjects, and was analyzed improperly (Szmigielski et al. 2001).

Several studies have investigated the risk of breast cancer in relation to RF exposure. A cohort study of radio and telegraph operators in Norwegian merchant ships by Tynes et al (Tynes et al. 1996) found a relative risk of breast cancer of 1.5 (95% confidence limits: 1.1 – 2.0), based on 50 cases in women working in this occupation and stronger for women aged 50 and above (2.6 (1.3 – 5.5)). An elevated relative risk found also for endometrial cancer suggests that reproductive and hormonal factors (for which full adjustment could not be made), not RF, may have been responsible for the raised breast cancer risk. A large case-control study based on job titles from death certificates in the US found no trend in risk of breast cancer in relation to probability or to level of occupational RF exposure (Cantor et al. 1995). A case-control study in the US of men with breast cancer found an odds ratio of 2.9 (0.8 – 10) in radio and communication workers (Demers et al. 1991), based on 7 cases in exposed men, and with a low response rate in controls. A study of US embassy personnel with potential RF exposure found 2 breast cancers with 0.5 expected (Goldsmith 1995). Other studies of male (Groves et al. 2002) and female (Lagorio et al. 1997; Morgan et al. 2000) breast cancers, with few cases, did not report increased risks. The available data are insufficient to reach any conclusion on whether RF exposure is related to breast cancer risk, but the results of Tynes et al. (Tynes et al. 1996) do support continued evaluation of the possibility.

Testicular cancer was considered in a US case-control study (Hayes et al. 1990). A significantly raised risk was found for self-reported occupational exposure to microwave and other radio waves (0R 3.1) but not for self-reported radar exposure nor for radar or other microwave exposure assessed by an occupational hygienist based on job history. A cluster of testicular cancer (observed/expected ratio = 6.9) was reported in 6 police officers in Washington State, US, who routinely used hand-held traffic radar guns (Davis and Mostofi

1993). In a large US Navy cohort with radar exposure, testicular cancer mortality was lower than expected (SMR 0.6 (0.2 – 1.4), n = 5) in the group with potential for high exposure (Groves et al. 2002).

Ocular melanoma was associated with self-reported exposure to microwaves (excluding domestic microwave ovens) or radar (0R 2.1 (1.1 – 4.0)) in a case-control study (Holly et al. 1996). Stang et al. (Stang et al. 2001) found a raised risk of ocular melanoma in subjects with self-reported occupational exposure for at least 6 months and several hours per day to RF (14% of cases, 10% of controls) and for occupational exposure several hours per day to radio sets (0R 3.3 (1.2 – 9.2)). There was no relation of risk to duration of this exposure, however, and risk was not raised for radar exposure (0R 0.4 (0.0 – 2.6)). The study was small, and combined subjects from two different study designs.

A nested case-control study of electrical utility workers in Quebec and France thought to be exposed to pulsed electromagnetic fields found a significant excess of lung cancer (Armstrong et al. 1994) and a dose-response gradient with increasing cumulative exposure. Adjustment for crude indicators of smoking and other factors left the results little changed. In an attempt to address a similar exposure in a cohort of US electric utility workers, limited due to the ill-defined agent addressed in the original study, no increased risk of lung cancer was found (Savitz et al. 1997). No other studies of RF have reported associations with lung cancer (Groves et al. 2002; Lagorio et al. 1997; Milham 1985; Milham 1988; Morgan et al. 2000; Muhm 1992; Szmigielski 1996; Szmigielski et al. 2001; Tynes et al. 1996).

In conclusion, there is no cancer site for which there is consistent evidence, or even an individual study providing strong evidence, that occupational exposure to RF affects risk.

The quality of information on exposure has generally been poor, however, and it is not clear that the heterogeneous exposures studied should be combined in aetiological studies. This, combined with imprecision and methodological limitations, leave unresolved the possibility of an association between occupational RF and cancer.

Other outcomes

Adverse Reproductive Outcomes

A wide range of potential reproductive consequences of RF exposure have been investigated (Table 5), with a focus on exposures of physiotherapists to therapeutic short wave diathermy (typically 27.12 MHz). Depending on the type of equipment used and the location of the operator in relation to the equipment, substantial peak exposures can occur (Larsen and Skotte 1991). Many of the studies analyzed levels of exposure, on the basis of duration of work and type of equipment used (shortwaves or microwaves).

There are isolated suggestions of an association between RF exposure and delayed conception (Larsen et al. 1991), spontaneous abortion (Ouellet-Hellstrom and Stewart 1993; Taskinen et al. 1990), stillbirth (Larsen et al. 1991), pre-term birth following exposure to fathers (Larsen et al. 1991), birth defects in aggregate (Larsen 1991), and increased male to female sex ratio (Larsen et al. 1991). Almost always, however, either the finding was not corroborated in other studies of comparable quality or there are no other studies available. The evidence is strongest for spontaneous abortion (based on two independent studies with some support). Potential confounding by other aspects of work activity (e.g., physical exertion) needs to be considered, however.

Semen parameters have been examined among men with varying forms of military exposure to microwaves and radar (Table 5). Three of these studies found reductions in sperm density, (Hjollund et al. 1997; Lancranjan et al. 1975; Weyandt et al. 1996), with variable results for other semen parameters, but one did not report such an association (Grajewski et al. 2000; Schrader et al. 1998). Several of these reports were based purely on volunteers with no attempt to sample from a defined population (Lancranjan et al. 1975; Schrader et al. 1998; Weyandt et al. 1996), and those that did provide information about response proportions (Grajewski et al. 2000; Hjollund et al. 1997) had substantial non-response. However, given the well-known susceptibility of spermatogenesis to even subtle heating, the possibility of reduced fertility in exposed men is reasonable to evaluate.

Overall, problems of exposure assessment temper any conclusions regarding reproductive outcomes, and no adverse effects of RF have been substantiated.

Cardiovascular Disease

Several methodologically weak studies from the Soviet Union addressed microwave exposure and acute effects on cardiovascular physiology (e.g., hypotension, bradycardia, tachycardia) as part of a set of ill-defined conditions (Jauchem 1997). Additional studies of considered symptoms among a range of potentially exposed groups including radar workers, pilots, radio broadcasting workers, and electronics industry workers. The variability in research methods, exposure characteristics, and outcome measures makes it difficult to draw conclusions: there are sporadic reports of symptoms among some groups of workers, but no obvious pattern is present.

Major clinical outcomes have been examined less frequently. In a mail survey of US physical therapists (Hamburger et al. 1983) men more highly exposed to microwave and shortwave radiation, based on indices including length of employment and frequency of treatments, tended to report a significantly greater prevalence of heart disease, with odds ratios of 2-3. Selective response to this survey must be considered among possible explanations for the associations that were observed. In US Navy veterans potentially exposed to radar (Groves et al. 2002) and in a cohort of nearly 200,000 Motorola workers (Morgan et al. 2000), heart disease SMRs were well below 1.0, and analyses of mortality (Groves et al. 2002), hospital admissions and disability compensation (Robinette et al. 1980) did not support greater risk with greater potential exposure. Other cohort studies reporting cardiovascular mortality have had small numbers (Lagorio et al. 1997; Muhm 1992).

Overall, the literature on RF and cardiovascular symptoms and disease provides little suggestion of an association, but is at too rudimentary a level to draw firm conclusions.

Cataract

Laboratory research indicates that the lens of the eye is highly sensitive to heat, and damage can occur from even a single acute exposure. Hence there is a potential mechanism for RF to lead to increased cataract incidence. Epidemiologic research has been limited, however, especially with regard to exposure assessment.

Based on hospital records of US military veterans (Cleary et al. 1965), men with cataracts were no more likely than men with other medical conditions to have been radar workers (OR 0.67, $p>0.10$). Age was adjusted using broad groupings, with little change to the result.

In two studies in the US military, ocular examinations were conducted on microwave-exposed and unexposed workers, without knowledge of exposure status by the examiner. In one (Cleary and Pasternack 1966) a tendency towards increased minor lens changes was found among exposed workers, characterized as the equivalent of 5 years advanced ageing in the exposed compared with unexposed workers around age 60. In the other (Shacklett et al. 1975), prevalence of lens opacities was similar in exposed and unexposed individuals matched on age.

In an Australian study of workers who built and maintained radio and television transmitters, compared with unexposed workers from the same geographic regions (Hollows and Douglas 1984), posterior subcapsular opacities were in excess in exposed workers (borderline significant) but nuclear sclerosis prevalence was similar in exposed and unexposed workers. It was not specified whether evaluators were aware of exposure history. Exposures were estimated to be from 0.08 to 3956 mW/cm^2, with brief, intense exposures thought to be quite common.

The study designs above are limited with respect to exposure assessment and selection of unexposed workers. Solar radiation exposure, a known risk factor for cataracts, was not considered and could have differed between RF exposed and unexposed workers. Not all of the opacities were of direct clinical importance, but they would be pertinent to a pathway that could lead to cataract later in life. The plausibility of a causal relation supports more extensive investigation.

Review of studies on environmental exposure from transmitters

The primary concern with transmitters has been with cancer risk among populations who live in proximity to transmitters, including those that are used for transmitting radio, television, microwave, and cellular telephone communications. There is a long history of public concern and resistance to the siting of such antennas, for reasons involving aesthetics and property values, as well as health concerns. Much of the research has been conducted in response to such concerns, either based solely on the exposure source or on a perceived cancer cluster among persons living in the vicinity.

The studies of which we are aware are listed in Table 6 together with some fundamental characteristics and major findings.

The first study (Selvin et al. 1992), in San Francisco was focused on statistical analysis of spatial data and the results are not reported according to standard epidemiologic practice and do not include relative risk estimates. The source of exposure was a large TV antenna, and the three statistical methods considered in the paper all showed that the pattern of cancer incidence was essentially random with respect to the antenna. A case-control study based on an apparent cluster of childhood leukaemia (Maskarinec et al. 1994) was prompted by an observation of an unusually large number of childhood leukaemia cases in a region of Hawaii. There were 12 leukaemia cases, and the odds ratio for having lived within 2.6 miles of the radio antennas before diagnosis was 2.0 (0.06 – 8.3). Hocking et al. (Hocking et al. 1996) compared cancer incidence in three municipalities immediately surrounding three TV transmitters in northern Sydney to the cancer incidence in six adjacent municipalities, estimating power densities from information on commencement of service of each

transmitter, power and frequency band. For leukaemia incidence in adults they found a relative risk of 1.2 (1.1-1.4) for the inner three municipalities compared with the surrounding municipalities. Their highest relative risk, 1.7 (1.1-2.5), was for the subcategory *other leukaemia*. For childhood leukaemia they observed a relative risk of 1.6 (1.1-2.3). Neither for adults nor for children were there any risk elevations for brain tumour.

Dolk et al. (Dolk et al. 1997) reported on an apparent cluster of leukaemia and lymphomas near a UK radio and TV transmitter at Sutton Coldfield. The study area was defined as a 10 km radius circle around the transmitter. Ten bands of increasing distance from the antenna were defined as the basis of testing for declining incidence with increasing distance. The relative risk of adult leukaemia within 2 km was 1.8 (1.2-2.7) and there was a statistically significant decline in risk with increasing distance from the antenna. In children under age 15 years there were 2 cases compared with 1.1 expected within the 2 km radius circle. The authors concluded that there was an excess risk of adult leukaemia in the vicinity of the transmitter.

A second investigation (Dolk et al. 1997) with a similar design to the first one was extended to include 20 high power TV and FM radio transmitters. Inside the 2 km radius circle the O/E ratio for adult leukaemia was 0.97 (0.78 – 1.2) and for childhood leukaemia 1.1 (0.61-2.1). Thus these results gave no more than very weak support to the original results.

McKenzie et al. (McKenzie et al. 1998) re-examined the Sydney results discussed above. They found that the excess risk reported by Hocking et al (Hocking et al. 1996) was mainly limited to one local government area within the studied region.

The Sutton Coldfield results have also been followed up by another group (Cooper et al. 2001). They used more recent cancer data to reanalyze cancer incidence around the transmitter and found considerably weaker results than the original.

An Italian study occasioned by local concerns investigated leukaemia incidence in children and leukaemia mortality in adults within a 10 Km circle around the Vatican radio station (Michelozzi et al. 2002). The station consists of numerous transmitters with different transmission powers ranging from 5 to 600 kW and with different frequency ranges. In adults of both sexes taken together the SMR within 2 km of the station was 1.8 (0.3-5.5) based on 2 cases. Stone's test for trend in rates over successive 2 km bands around the station gave a p-value of 0.14. The excess risk and the trend were essentially confined to males. In children, the SIR for those living within the 2 km radius circle was 6.1 (0.40-28) based on one case. Elevated rates were observed for all cumulative bands up to 10 km but all had wide confidence intervals and the total number of cases within the 10 km radius circle was 8. The Stone test for trend was reported as p=0.004. No systematic RF measurements have been made in the area and the epidemiologic analyses are based on the simplistic proxy, distance from the source. The numbers of cases were small, especially for children, which precludes firm conclusions. For adults the results were inconsistent with the risk elevations largely confined to males.

Discussion

The research on community exposures to radiofrequency fields and cancer gives a very weak test of the possibility of a relation. Diverse exposure sources, poorly estimated population exposures, small numbers of cases, and selective investigation in response to cluster concerns have resulted in a literature that is of limited value. Despite apparent positive relations

between proximity and leukaemia incidence in some analyses (Hocking et al. 1996; Michelozzi et al. 2002), the results have not been consistent within or between studies, and do not show relations to RF exposure levels . It seems to us that a prerequisite for a new generation of informative studies to emerge is the use of an RF meter.

Some of the concern about health risks from living near transmitters is directed toward symptoms such as fatigue, sleep disturbances, and frequent headaches. It may be tempting to address such issues in a cross-sectional study of people living near transmitters, in which subjects are asked to report their symptoms. Indeed, such studies have been done (Navarro et al. 2003; Santini et al. 2002; Santini et al. 2003). However, this is a design in which exposure is poorly characterized and reporting bias with respect to symptoms is of concern. Experimental designs easily overcome these biases and thus would be preferable, although they have their own limitations such as difficulty in practice in detecting effects present in a small percentage of a population or when the effect is not immediate. In these latter situations, an observational study would be the design of choice, but only if a design was found that avoided reporting bias.

Review of studies on mobile phone use

Most studies of association between cancer and mobile phone use have evaluated the risk of brain tumours and acoustic neuromas (Table 7), though in a few instances the risks of other tumours have been explored. Also studies of symptoms in relation to mobile phone use have been conducted (Table 8). The first case-control study of brain tumours was conducted in Sweden (Hardell et al. 2001; Hardell et al. 2000; Hardell et al. 1999) and included adult cases diagnosed in two regions in Sweden between 1994 and 1996 and still alive, with two controls

per case matched for region of residence. Details of intensity and duration of mobile phone use, preferred side (ear) of use, and whether phones were analogue or digital, and handheld or hands-free, were gathered by postal questionnaire followed by telephone interview (Hardell et al. 1999). 209 cases (about a third of the malignant cases occurring in the study geographical area in the period (Ahlbom and Feychting 1999)) took part along with 425 controls (a reported 91% response rate – extraordinarily high for a contemporary population-based study). Originally no association of phone use with brain tumours was found (Hardell et al. 1999), though later re-analysis of side of use in relation to tumour site suggested a possible relationship (Hardell et al. 2001). A second larger study a few years later by the same authors (Hardell et al. 2002; Hardell et al. 2003) was similar in design to the first. It involved 1303 living cases (half of all brain tumours diagnosed 1997 – 2000) and their controls. Cumulative phone use for over 85 hours, 10 years before case diagnosis, gave ORs for brain tumours of 1.9 (1.1-3.2) and 3.0 (0.6-14.9) respectively for analogue and cordless phones, but not raised for digital. There was no adjustment for confounding variables. Ipsilateral use of analogue phones was related to temporal tumours (OR 2.5 (1.3-4.9)), and analogue phone use was associated with acoustic neuroma (OR 3.5 (1.8-6.8)) (Hardell et al. 2002; Hardell et al. 2003).

Muscat et al conducted two hospital-based case-control studies in the USA, one of malignant brain tumours (Muscat et al. 2000), the other of acoustic neuroma (Muscat et al. 2002), both using the same ascertainment and data collection procedures (Table 7). The first study included 469 cases of brain cancer (70% response rate), and 422 matched controls with a variety of malignant and benign conditions from the same hospitals (90% response rate). Information about mobile phone use was obtained by standard interview (of proxies for 9% of cases and 1% of controls). No raised risks were seen relating to frequency or duration of use, nor for site or histologic subtype of brain cancer. An excess of brain cancer was found on the

same side of the head as reported phone use among 41 cases with assessable data (p = 0.06), compared with a deficit on the side of mobile phone use for tumours specifically located in the temporal lobe (p = 0.33). In the acoustic neuroma study, 90 cases were compared with 86 controls, and no associations were seen with level or laterality of phone use.

In another US hospital-based case-control study (Inskip et al. 2001), interview data were obtained from 782 cases with brain tumours (92% response rate; via proxies for 16% and 3% of glioma and acoustic neuroma patients respectively) and 799 matched hospital controls with non-malignant conditions (88% response; 3% by proxy). Results adjusted for potential confounders showed no association between cumulative use of mobile phones (mainly analogue) and brain tumour overall or by histological subtype or anatomical location.

Subscription records of national network providers were used to characterise mobile phone users in a Finnish case-control study (Auvinen et al. 2002). All people (398) diagnosed with brain tumours in 1996, ascertained from the National Cancer Registry, were matched with 5 controls per case drawn from the national population register (Table 7). The OR for brain tumours with ever-subscription to phones was 2.1(1.3 - 3.4) for analogue phones and 1.0 for digital, and the OR for glioma was 1.5 (1.0 – 2.4) for any phone subscription. The average duration of subscription was 2-3 years for analogue phones and less for digital. Adjusting for potential confounders did not alter results. No information was available about the frequency or duration of calls or about corporate subscriptions.

Of two cohort studies, an early US study (Dreyer et al. 1999; Rothman et al. 1996) analysed one year of follow-up of mortality in a cohort of 285,561 non-corporate users of mobile phones with at least 2 billing cycles from two US carriers. Mortality was ascertained from the

National Death Index. No relation was found between mortality from brain cancer and the use of handheld versus hands-free phones, based on only six cases. The overall mortality of the cohort was less that in the general population. The second cohort study was in Denmark (Johansen et al. 2002) and comprised 420,095 private cellular network subscribers (80% of all subscribers), with average follow-up for analogue and digital subscribers of 3.5 and 1.9 years respectively. Standardised incidence ratios comparing cancer rates in phone users with national rates allowing for sex, age and period, showed no relation to risk of brain and nervous system cancers (SIR 0.95 (0.81 – 1.2)) and reduced risk of smoking related-cancers. Risks did not vary by age at, or time since, first subscription, phone type or tumour location. Again no information was available about the frequency or duration of calls or about corporate subscriptions.

Regarding other head and neck cancers, no association with parotid gland tumours (34 cases) was seen in the Finnish case-control study (Auvinen et al. 2002), or in the Danish cohort study (Johansen et al. 2002). A mixed population and hospital-based case-control study of uveal melanoma (Stang et al. 2001) included 118 cases and 475 controls. Occupational exposure to mobile phones for several hours a day for 6 months or more assessed by interview gave a raised OR (4.2 (1.2 – 15)), reflecting the result in the hospital-based participants (OR 10). There was no raised risk of uveal melanoma, however, in the Danish mobile phone user cohort (Johansen et al. 2002). Finally, leukaemia was assessed in both cohort studies, but no relation with phone use was found.

The first report from the multicentre Interphone study, a very large, multicenter international case-control study, has recently been published. This report from the Danish component

focused on acoustic neuroma and was negative; however, the number of long term users was small (Christensen et al. 2004).

Subjective symptoms, including tinnitus, headache, dizziness, fatigue, sensations of warmth, dysaesthesia of the scalp, visual symptoms such as flashes, memory loss and sleep disturbance have been investigated in relation to mobile phone use (Chia et al. 2000; Oftedal et al. 2000; Sandstrom et al. 2001). See Table 8 for details. As discussed above in relation to transmitter studies, such research is highly susceptible to recall bias and for completeness we have added a table(Table 9) with experimental studies on mobile phone use and symptoms.

Discussion

Handheld mobile phones were not used regularly until the 1990s, so published studies at present can only assess relatively short lag periods before cancer manifestation. The relevant lag periods are unknown. Furthermore, even in the large Danish study (Johansen et al. 2002), long-term (15 years) subscribers to analogue phones comprised only a small proportion of users.

Another issue relates to choice of study population. No study populations to date have included children, yet children are increasingly heavy users of mobile phones and they are potentially highly susceptible to harmful effects (although some of these effects might not manifest until adulthood). So far study populations have been ascertained from population registers in Nordic studies, hospital in-patients in the US case-control studies, and cellular network private subscribers in the two cohort studies and the Finnish study. While the population-based studies should have avoided the selection biases inherent in the hospital

based studies, this was not so in population-based case-control studies of prevalent living cases with low participation rates (Hardell et al. 2002; Hardell et al. 1999) since inter alia those with high grade tumours tend to be excluded. While rapid recruitment of incident brain tumour cases was facilitated in the hospital-based studies, loss due to death was still greater for malignant than benign tumours as reflected in differential proxy response rates by tumour type (Inskip et al. 2001), and there is a weakness in using hospital controls with a variety of conditions of unknown relationship to mobile phone use.

Differential recall of mobile phone use among those with and without a cerebral tumour in case-control studies is a major potential source of bias, exacerbated by differential timing of data collection from cases and controls in the hospital studies. Reporting bias is also likely since presence of a brain tumour may distort both memory and hearing and because the use of proxy respondents was more common for cases than controls. Relying on private cellular network subscription as a measure of mobile phone use would also have resulted in substantial misclassification because subscribers bear only a modest relation to users (Funch et al. 1996) and because corporate users were either excluded or included in the unexposed group. Until there is some objective measure of RF exposure, or at least validation of self-reported records, the validity of self-reported indices of phone use e.g. average minutes of use per day (Hardell et al. 2002; Inskip et al. 2001) or minutes/hours per month as indicators of RF exposure, remains unknown.

Overall, while occasional significant associations between various types of brain tumour and analogue mobile phone use have emerged (often seen after multiple testing), no single association has been consistently reported across population-based studies. The timing of epidemiological studies and the lack of knowledge about actual RF exposure to the brain from

mobile phone use to date (Ghandi et al. 1999) militate strongly against current ability to detect any true association. Thus current evidence is inconclusive regarding cancer risk following heavy RF exposure from mobile phones. Similarly the studies of symptoms to date do not suggest that a single exposure to RF from a mobile phone results in immediately identifiable symptoms, but there are no adequate data available about the symptomatic effects of mobile phone use, especially among people who claim hypersensitivity to RF.

General conclusions and recommendations

Results of epidemiological studies to date give no consistent or convincing evidence of a causal relation between RF exposure and any adverse health effect. On the other hand, these studies have too many deficiencies to rule out an association.

A key concern across all studies is the quality of assessment of RF exposure, including the question of whether such exposure was present at all. Communication sources have increased greatly in recent years, and there is continuing change in the frequencies used and the variety of applications. Despite the rapid growth of new technologies using RF, little is known about population exposure from these and other RF sources and even less about the relative importance of different sources. Certain studies that are currently under way have made serious attempts to improve exposure assessment, based on attempts to learn more about determinants of RF exposure levels. A key element in improving future studies would be the use of a meter that monitors individual exposure. In the absence of information on what biological mechanism is relevant, if any, it is unclear what aspect of exposure needs to be captured in epidemiological studies. Ideally, the dose needs to be assessed not just as external field intensity, but also as cumulative exposure, as well as SAR, for specific anatomical sites.

The need for better exposure assessment is particularly strong in relation to transmitter studies, because the relation between distance and exposure is very weak. There is no point in conducting such studies unless it has been established that exposure levels vary substantially within the study area, and measurements of these RF levels are available. In the future, methods need to be developed to infer exposure based on some combination of knowledge regarding the sources of exposure, the levels of exposure, and location of people in relation to those sources, ideally informed by selective measurements.

Although the likelihood is low that fields emanating from base stations would create a health hazard because of their weakness, this possibility is nevertheless a concern for many people. To date no acceptable study on any outcome has been published on this. On the one hand, results from valid studies would be of value in relation to a social concern; on the other hand, it would be difficult to design and conduct a valid study, and there is no scientific point in conducting an invalid one.

Another general concern in mobile phone studies is that the lag periods that have been examined to date are necessarily short. The implication is that if a longer lag period is required for a health effect to occur, the effect could not be detected in these studies. Only in the few countries where mobile phones were introduced very early has it been possible to look at usage ten years ago or more. Much longer lag periods have been examined for occupational RF exposures, however. The published studies include some large occupational cohorts of good design and quality, except that there has been poor assessment of the degree of RF exposure, which render the results difficult to interpret.

The majority of research has focused on brain tumours and to some extent on leukaemia. However, because the RF research questions are not driven by a specific biophysical hypothesis but rather by a general concern that there are unknown or misunderstood effects of RF fields, studies on other health effects may be equally justified. Examples are eye diseases, neurodegenerative diseases, and cognitive function. Given the increase in new mobile phone technologies, it is essential to follow various possible health effects from the very beginning and for long periods, since such effects may be detected only after a long duration, due to the prolonged latency period of many chronic diseases. Thus, research is needed to address long-term exposure, as well as diseases other than those included in the ongoing case-control studies.

Another gap in the research is children. No study population to date has included children, with the exception of studies of people living near radio and TV antennas. Children are increasingly heavy users of mobile phones. They may be particularly susceptible to harmful effects (although there is no evidence of this), and they are likely to accumulate many years of exposure during their lives.

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TABLE 1. Cohort studies of risk of cancer in relation to occupational or hobby RF exposure : description of studies

Authors, year	Occupational group	Sex	No. of subjects	Measure of exposure	Outcome
Milham 1988	Amateur radio operators	Male	67,829	Hobby title	Mortality
Garland et al. 1990	Navy personnel: electronics technicians, aviation electronics technicians, fire control technicians[a]	Male	-[b]	Job title	Incidence
Muhm et al. 1992	Electromagnetic pulse test workers	Male	304	Job title	Mortality
Tynes et al. 1996	Radio & telegraph operators on merchant ships	Female	2,619	Measures in radio rooms of 3 ships	Incidence
Szmigielski 1996[c]	Military career personnel	Male	128,000 total[d] 3,700 exposed[d]	Military health records; representative exposure levels given, based on measurements (no. not stated)	Incidence
Szmigielski et al. 2001	Military career personnel	Male	124,500 total 3,900 exposed		
Lagorio et al. 1997	Dielectric RF heat sealer operators	Female	481	Unclear – stated that >10W/m^2 'frequently exceeded'	Mortality

Morgan et al. 2000	Motorola employees	56% male 44% female	195,775 total 24,621 exposed	Job title, with expert assessment (not measured)of usual exposures	Mortality
Groves et al. 2002	Navy personnel with potential radar exposure	Male	40,581 total 20,021 high exposure	Job title, plus expert assessment on potential for high exposure, and information on type and power of radar units	Mortality
Lilienfeld cited by Goldsmith 1995	US embassy personnel	Males & Females	Not stated	Moscow embassy service	Mortality

[a] We have extracted from the published paper data on those jobs stated by Groves et al (2002) to have greatest RFR exposure.
[b] Not stated.
[c] Not strictly a cohort study – there does not appear to be any follow-up. Design appears to be calculation of annual rates, based on annual incidence and counts of employed population, and then averaging of these rates.
[d] "Mean count each year ". Presumably many but not all of the personnel will have been the same individuals from year to year of the study.

TABLE 2. Cohort studies of risk of cancer in relation to occupational RF exposure : results for brain tumour and leukaemia

Author, year	Type of analysis	Brain tumour		Leukaemia		Comment
		n	Relative risk (95% CI)	n	Relative risk (95% CI)	
Milham 1988	SMR, cohort *cf* general	29	1.4 (0.9 – 2.0)	36	1.2 (0.9 – 1.7)	In a sample, 31% of subjects population worked in EMF-exposed occupations. Analyses by license class, a proxy for duration of licensing, showed no consistent trend in risk. (Milham 1988b)
Garland et al. 1990	SIR, cohort *cf* general population					
	Electronics techn	-[a]		5	1.1 (0.4 – 2.5)	
	Aviation tech.	-[a]		<3	0.3 (0.0 – 1.9)	
	Fire control tech.	-[a]		<3	0.5 (0.0 – 2.5)	
Muhm et al. 1992	SMR, cohort *cf* general population, underlying cause	0	-	1	4.4 (0.1 – 24.3)	One of the leukaemia cases may have been allocated to this work because of his leukaemia
	SMR, cohort *cf* general population, mentioned cause	0	-	2	7.7 (0.9 – 28.0)	
	SIR, cohort *cf* general population	-	-	2	5.4 (0.7 – 19.7)	
Tynes et al. 1996	SIR, cohort *cf* general population	5	1.0 (0.3 – 2.3)	2	1.1 (0.1 – 4.1)	
Szmigielski 1996[a]	Average crude incidence rate in exposed *cf* average crude rate in unexposed.	-[a]	1.9[b] (1.1 – 3.5)	-[a]	7.7[c] (-[a])	Poorly conducted and reported study. Apparently more exposure data sources for cases than controls.

Szmigielski et al. 2001		7	2.7[b] (p<0.01)	19	6.5[c] (p<0.01)	'Expected' rates in the 1996 paper appear to be incorrect, according to the Royal Society of Canada (The Royal Society of Canada 1999) Significant excesses reported for several cancer sites not seen in other studies, and for cancer overall, suggesting possible bias. Analyses of risk in relation to exposure level presented only for total cancer, not specific cancer sites.
Lagorio et al. 1997	SMR, cohort *cf* general population	1	10	1	5	Potential confounding by chemical exposures. Losses to follow-up treated as alive to end of study period.
Morgan et al. 2000	SMR, exposed workers *cf* general population	17	0.5 (0.2 – 1.1)	21	0.8 (0.4 – 1.4)	No duration-response trend.
	Rate ratio exposed *cf* unexposed in cohort, cumulative exposure					
	None	34	1.0	66	1.0	
	<median	7	1.0 (0.4 – 2.2)	8	0.6 (0.3 – 1.3)	
	≥median	10	0.9 (0.4 – 1.9)	13	0.6 (0.3 – 1.0)	
Groves et al. 2002	SMR, overall cohort *cf* general population	88	0.9 (0.7 – 1.1)	113	1.0 (0.8 – 1.2)	Significant raised risk for nonlymphocytic leukaemia in high exposure cohort, but only raised in one of 3 high-exposure occupations.
	SMR, high exposure cohort *cf* general population	37	0.7 (0.5 – 1.0)	69	1.1 (0.9 – 1.4)	
	Relative risk, exposed *cf* unexposed in cohort	37/51	0.6 (0.4 – 1.0)	69/44	1.5 (1.0 – 2.2)	

Lilienfeld cited by Goldsmith 1995[d]	Observed and expected, but source of latter unclear	Adults: 2/1.9 Children: 0/-	2/2.0 2/4.0	Data also presented for other US embassies in Eastern Europe, but unclear whether they were exposed. Both brain tumours and one leukaemia in a child were in dependents who lived out of the embassy.

[a] No data published. For Szmigielski it is implied that there were 2-3 brain tumours and X leukaemias in the exposed group, in which case we imply that the CI for brain tumour is incorrect.
[b] Nervous system.
[c] Calculated from data in the paper.
[d] Study not published by Lilienfeld, and too little information given in précis by Goldsmith for understanding or evaluation of the methods. Small numbers of cancers, and several of the cancers occurred in persons who lived out of the embassy (i.e. presumably were in the embassy little of the time, especially children). Breast cancer in employees 2 observed, 0.5 exp. Cancers of female genitalia 4 observed, 0.8 expected. Exposures estimated to range from 5 – 18μ W/cm^2 – basis of estimate not stated.

TABLE 3. Case-control studies of risk of brain tumour and leukaemia in relation to occupational RF exposure.

Authors, year	Sources of cases and controls[a]	Measure of exposure	Exposure data collection method	Mortality or incidence	Nos. of cases/ controls	Type of analysis	Results	
							Brain tumour OR (95% CI)	Leukaemia OR (95% CI)
Thomas et al. 1987	Cases: death certificates Controls: death certificates for deaths from other causes, except epilepsy, stroke, suicide, homicide.	Job title & industry	Interview with relatives	Mortality	435/386	Odds ratio *cf* never occ. exposed	1.6 (1.0 – 2.4)	-
Amstrong et al. 1994	Electrical utility workers (nested case-control)	Job exposure matrix based on 1 week meter measurements >1000 workers, assessing exposure to pulsed electro-magnetic fields.	Company records at 5-20 MHz[b] for	Incidence	84/325 95/374	Odds ratio for ≥median exposure Odds ratio for ≥90th percentile Odds ratio for ≥median exposure Odds ratio for ≥90th percentile	0.8 (0.5 – 1.5)[c] 1.9 (0.5 – 7.6)[c] - -	- - 0.7 (0.4 – 1.2) 0.8 (0.2 – 3.4)
Grayson 1996	USAF (nested case-control)	Job title & whether reports of incidents of high exposure for each job title	Military records	Incidence	230/920	Odds ratio *cf* never-exposed	1.4 (1.0 – 1.9)	-

[a] All studies restricted to men.
[b] But it was subsequently found that the meters also responded to fields of 150 and 300 MHz and to radio transmissions.
[c] Malignant brain tumours.

TABLE 4. Analyses of routinely collected data on brain tumour and leukaemia risk in relation to occupational RF exposure.

Author, year	Type of analysis	Exposed group[a]	Comparison cohort/ control group	Mortality or incidence	Brain tumour		Leukaemia	
					n[b]	RR (95% CI)	n[b]	RR (95% CI)
Wright et al. 1982	Proportional incidence	Radio & TV repairmen.	All other cancers	Incidence	-[c]		1	1.2 (-[c])
		Telephone linesmen.			-[c]		2	3.1 (-[c])
Calle & Savitz. 1985	Proportional mortality	Radio & telegraph operators.	All causes of death	Mortality	-[c]		6	2.3 (-[c])
		Radio & TV repairmen.			-[c]		3	0.9 (-[c])
Lin et al. 1985,	Case-control	Electric & telephone linemen, servicemen.	Non-cancer deaths	Mortality	27			(-[c])
Milham 1985	Proportional mortality	Radio & telegraph. operators.	All causes of deaths	Mortality	1	0.4 (-[c])	5	1.0 (-[c])
		Radio & TV repairmen.			2	0.6 (-[c])	7	1.8 (-[c])
Pearce et al. 1989	Case-control	Radio & TV repairmen.	All other cancers	Incidence	-[a]		2	7.9 (2.2 – 28.1)
Tynes et al, 1992	Cohort	Radiofrequency exposed occupations.	Economically active males	Incidence	3	0.6 (0.1 – 1.8)	9	2.8 (1.3 – 5.4)

[a] All studies are of males. Exposure assessment for all is based solely on job title, with no measures of exposure.
[b] No. in exposed group.
[c] No data published.

TABLE 5. Summary of literature on radiofrequency exposure and reproductive health outcomes

Outcome	Reference	Geographic setting	Population size & source	Results: Exposure & outcome
Semen parameters				
	Lancranjan et al. 1975	Romania	Microwave exposure (31) vs. controls (30)	Sperm count: 50 (Exp), 60 (Ctl) million/ml Motility: 36% (Exp), 54% (Ctl)
	Weyandt et al. 1996	US	Military intelligence (20) vs. controls (30)	Sperm density: 13 (Exp), 35 (Ctl) Percent normal: 69 (Exp), 73 (Ctl) Percent motile: 32 (Exp), 43 (Ctl)
	Hjollund and Bonde 1997	Denmark	Military: missile operators (19), other (489)	Sperm density: 40 (Exp), 62 (Ctl) Immotile %: 52 (Exp), 33 (Ctl) Percent normal: 61 (Exp), 68 (Ctl)
	Schrader et al. 1998	US (Texas)	Military: radar operators (33), controls (103)	Sperm density: 29 (Exp), 32 (Ctl) Percent normal: 46 (Exp), 42 (Ctl) Percent motile: 46 (Exp), 45 (Ctl)
	Grajewski et al. 2000	US (Maryland)	RF heater operators	Sperm density: 47 (Exp), 45 (Ctl) Sperm count: 73 (Exp), 93 (Ctl) Motile (%): 67 (Exp), 52 (Ctl) Normal morphology: 81 (Exp), 79 (Ctl)
Fertility				
	Larsen et al. 1991	Denmark	Physiotherapists 49 time to pregnancy over 6 months	TWA exposure & TTP >6 months RR = 1.0, 0.8 (0.2-2.2), 1.7 (0.7-4.1):

Spontaneous abortion				
	Taskinen et al. 1990	Finland	Physiotherapists 204 Spontaneous abortions	SAb <=10 Deep heat 1.0,1.3,0.7, Shortwaves 1.0,1.2,0.7, Microwaves 1.0,0.7 SAb >10 Deep heat 1.0, 1.3, 2.6, Shortwaves 1.0,2.5,2.4; Microwaves 1.0,2.4
	Larsen et al. 1991	Denmark	Physiotherapists 146 Spontaneous abortions	TWA Exposure & Sab: RR = 1.0, 1.0 (0.5-1.8), 1.4 (0.7-2.8)
	Ouellet-Hellstrom and Stewart 1993	US	Female physical therapists 1664 Spontaneous abortions	Microwave Diathermy Exposures/mo. RR=1.0, 1.1(0.8-1.4), 1.5 (1.0-2.2), 1.6 (1.0-2.6) Shortwave Diathermy Exposures/mo: RR=1.0, 1.2 (1.0-1.5), 1.1(0.9-1.4), 0.9 (0.6-1.2)
Stillbirth				
	Larsen et al. 1991	Denmark	Physiotherapists 17 perinatal deaths	TWA Exposure & Perinatal Death: RR = 1.0, 1.5 (0.3-5.3), 2.9 (0.6-10.7)
Preterm Birth				
	Larsen et al. 1991	Denmark	Physiotherapists 37 male, 45 female	TWA Exposure & Preterm Birth: Male: RR=1.0, 1.4 (0.4-4.7), 3.2 (0.7-13.2) Female: RR=1.0, 0.9 (0.4-2.1), 0.9 (0.3-2.8)
Low Birth Weight				
	Larsen et al. 1991	Denmark	Physiotherapists 15 male, 24 female	TWA Exposure & Low Birthweight: Male: RR=1.0, 0.0, 5.9 (1.0-28.2) Female: RR=1.0, 1.2 (0.4-3.3), 0.7 (0-3.2)
	Guberan et al. 1994	Switzerland	Physiotherapists 11 male, 14 female	No association with shortwaves (RR not reported)

Birth Defects				
	Logue et al. 1985	US	Physical therapists (male) 192 birth defects	Observed: expected range - "appears to be higher than expected"
	Taskinen et al. 1990	Finland 51 birth defects	Physiotherapists	Deep heat 1.0, 2.4 (1.0-5.3), 0.9 (0.3-2.7) Shortwaves 1.0, 2.7 (1.2-6.1), 1.0 (0.3-3.1) Microwaves 1.0, 0.5 (0.1-3.9)

TABLE 6. Summary of studies on transmitters and cancer

Reference	Source of exposure	Comparison	Endpoints	No. cases	Results	Setting	Comments
Selvin et al. 1992	MW antenna	Internal	Childh ca Childh leuk	123 52	Random pattern	San Francisco	Analysis of spatial data; no epi param
Maskarinec et al. 1994	LF radio (23.4 kHz)	<2.6 miles	Childh leuk	12	2.0: 0.06-8.3	Hawaii; case-control	SIR analysis on same cases: 2.09: 1.08-3.65
Hocking et al. 1996	TV antenna	Inner/ outer	All age leuk Childh leuk		1.24: 1.09-1.40 1.58: 1.07-2.34	Northern Sydney	8 –0.2μW/cm^2
Dolk et al. 1997 I	TV and FM radio	<2 km	Adult leuk	23	1.83:1.22-2.74	Sutton Coldfield	
Dolk et al. 1997 II	TV and FM radio	<2 km	Leukaemia	79	0.97: 0.78-1.21	All GB	
McKenzie et al.1998	TV antennas	Cont. μW/cm^2 model	Childh leuk			Sydney	Reanalysis of Hockings; concl. One LGA explains
Cooper et al. 2001	TV and FM radio	<2 km	All age leuk Childh leuk	20 1	1.32. 0.81-2.05 1.13: 0.03-6.27	Sutton Coldfield	Reanalysis, more timely cancer data
Michelozzi et al. 2002	Radio station	<6 km	Childh leuk Adult leuk	8 23	2.2: 1.0-4.1 1.2: .8-1.8	Vatican	

TABLE 7: Summary of studies of mobile phone use and risk of brain tumours**.**

Authors, year (study design)	Study population	Tumour type (numbers cases/ controls)	Exposure assessment	Phone type; Duration of use in controls	Phone ever-use RR (95% CI)
Hardell et al. 1999 (case-control)	Sweden. Cases: 20-80 yr. Controls: regional population registers, Uppsala-Orebro 1994-96, Stockholm 1995-96	All tumours (209/ 425) Acoustic neuroma	Recalled mobile phone use by questionnaire and interview.	Mainly analogue, 450 or 900 MHz; 16% >5 yr.	1.0 (0.7 – 1.4)[a] 0.8 (0.1 – 4.2)
Muscat et al. 2000 (case-control)	USA: Hospital inpatients, NY, Providence, Boston. Cases:18-80 yr ,1994-98. Controls: Malignant and non-malignant conditions.	Malignant brain tumour (469/ 422)	Recalled mobile phone use via interview	Mainly analogue 800 – 900 MHz; 5% >4 yr	0.9 (0.6 – 1.2)
Inskip et al. 2001 (case-control)	USA: Hospital inpatients, Boston; Phoenix; Pittsburgh.Cases:18+ yr, 1994-98. Controls: non-malignant conditions	All tumours (782/799) Glioma (489/799) Meningioma (197/ 799) Acoustic neuroma (96/799)	Recalled mobile phone use via interview	Mainly analogue 800 – 900 MHz; 8% >3 yr.	0.9 (0.7 – 1.1) 1.0 (0.7 – 1.4) 0.8 (0.5 – 1.2) 0.8 (0.5 – 1.4)
Muscat et al. 2002 (case-control)	USA: Hospital inpatients, New York. Cases:18+ yr, 1997-99. Controls: Non-malignant conditions.	Acoustic neuroma (90/86)	Recalled mobile phone use via questionnaire	Mainly analogue 800 – 900 MHz; 7% 3-6 yr.	0.9
Auvinen et al. 2002	Finland. Cases: 20-69 yr, 1996. Controls: National	All tumours (398/1986) Glioma (198/989)	Duration of private cellular	Analogue, average 2-3 yr subscription;	1.3 (0.9 – 1.8) 1.5 (1.0 – 2.4)

(case-control)	population register.	Benign (129/643) Salivary gland (34/170)	network subscription	digital, average <1 yr subscription	1.1 (0.5 – 2.4) 1.3 (0.4 – 4.7)
Hardell et al. 2002 (case-control)	Sweden Cases: 20-80 yr. 1997-2000. Controls: 4 regional population registers.	All tumours (1303/1303)	Recalled mobile phone use via questionnaire	Analogue 450 or 900 MHz, median 8 yr. Digital 1900 MHz, median 3 yr.	1.3 (1.0 – 1.6)[a] 1.0 (0.8 – 1.2)
Hardell et al. 2003 (case-control)		Acoustic neuroma (159/ 422)		Analogue Digital	3.5 (1.8 – 6.8) 1.2 (0.7 – 2.2)
Dreyer et al. 1999 (cohort)	USA. Subscribers of 2 large cellular networks. 1993. Cases: ≥20 yr deaths 1994	Malignant brain tumour (6)	Duration of subscription	Analogue. 1 yr follow-up	- -
Johansen et al. 2002 (cohort)	Denmark. Private cellular network subscribers, 1982-95. Cases: ≥18 years, 1982-96.	All tumours (154) Glioma (66) Menigioma (16)	Duration of subscription	Analogue (450 or 900 MHz) or digital. Up to 15 yr follow-up	SIR1.0 (0.8-1.1) 0.9 (0.7 – 1.2) 0.9 (0.5 – 1.4)
Christensen et al. 2004	Denmark Population-based case-control	Acoustic neuroma (106) Population controls (212)	-	-	0.90 (0.51 – 1.6)

[a] Analysed with a 1 year lag period discounted.

TABLE 8. Summary of studies of mobile phone use and symptoms.

Authors, year (study design)	Study population	Analyses	Exposure assessment	Outcome assessment	Results
Hocking 1998 (case-series)	Australians with symptoms on mobile phone use who responded to notice in medical journal or media publicity N=40	Description of type of symptoms reportedly due to mobile phone use	No formal assessment of amount or frequency of mobile phone use	Questionnaire about details of symptoms associated with mobile phone use	Most respondents reported unusual sensations affecting the head, such as dull pain, unpleasant warmth
Oftedal et al. 2000 (cross-sectional)	Swedish and Norwegian mobile phone users, selected from network operator registers. Only included people who used phone for job. N=10631	1. Number of respondents with any symptom attributed to mobile phones 2. Number of respondents who had taken steps to reduce symptoms	Not well described, but one table reports number of calls and calling time per day, suggesting reported in a questionnaire	Self-reported frequency of symptoms. Patient considered to have symptom if occurred at least once per week	1. 13% of participants in Sweden and 31% in Norway reported at least one symptom in connection with use of a mobile phone. Most common,warmth around ear. 22% of Norwegians and 7% of Swedes experienced symptom other than warmth. 2. 45% of people experiencing symptoms had taken steps to reduce them, such as reduced calling time, use of hands free kit, changing side phone used.
Sandstrom	Swedish and	1. Comparison of	Self-completed	Self-reported	1. OR among GSM cf NMT phones:

et al. 2001 (cross-sectional) .	Norwegian mobile phone users, selected from network operator registers. N=16,992	GSM versus NMT mobile phone users 2. Trends with increasing time of phone usage	questionnaire, variables; transmitter system, calling time per day and number of calls per day	frequency of range of symptoms. Participant consi-dered to have symptoms if occurred at least, once per week	No increased risk for any symptoms. GSM users at lower risk of warmth behind ear (OR: 0.64, 95% CI 0.51-0.80)or on ear (OR: 0.68, 95% CI 0.53-0.86). GSM users in Sweden at lower risk of headaches (OR: 0.73, 95% CI 0.56-0.95) and fatigue (OR: 0.8095% CI 0.65-0.99) 2. With increasing minutes of phone use there was an increased odds of reporting fatigue, headaches, warmth, burning and tightness at least once per week.
Chia et al. 2000 (cross-sectional)	Random sample of 635 households in housing estate in Singapore. 808 respondents (NB response rate less than 60%).	1. Prevalence ratio of headache in mobile phone users vs non-users 2. Association between minutes, phone use and headache	Interviewer-administered questionnaire. Purpose of study masked. Classified as MP user if used at least once/day .	Questionnaire concerning nature and severity of 'CNS symptoms' (headache, dizziness, warmth, tingling, visual disturbances). NB the frequency of headaches required before a respondent was classified as a headache sufferer was not specified.	1. 45% mobile phone users 3% experienced CNS problems. Adjusted prevalence ratio for headache among users cf non-users 1.31 (95% CI 1.00-1.70). No significant differences for any other symptoms. 2. Significant positive trend for increasing time spent on the mobile phone and prevalence of headache (p=0.04).

TABLE 9. Summary of experimental studies of mobile phone use and symptoms.

Authors	Participants	Exposure	Protocol source	Symptoms reported	Results
Hietanen et al. 2002 (experi-mental)	20 volunteer subjects, mean age 51 for women and 47 for men, all of whom classified them-selves as hypersensitive to RF fields	Analogue NMT phone, transmitting at 900MHz. 900 and 1800MHz GSM phones.	Phones mounted near but not touching subjects ear. 3 or 4 experimental sessions lasting 30 minutes each, one of which was a sham exposure (random order)	Subjects asked to describe symptoms experienced during exposure. Blood pressure, heart rate and breathing frequency monitored. Follow-up form used to measure symp-toms over subsequent days	19/20 participants reported symptoms during the tests. Compared with women during sham exposure, relative number of symptoms reported by female subjects during NMT exposure was 0.82, GSM 900 0.79, GSM 1800 0.72. Among men, number of symptoms during any RF exposure situtation was 0.85 compared with sham exposure.
Koivisto et al. 2001 (experi-mental)	48 volunteers, students at University of, Turku Finland. Mean age 26 years	GSM 900 phone	2 exposure sessions, one with mobile phone on and one with off Subjects blinded to whether phone was off or on. Half participants had phone on first and half off first	Questionnaire assessing symptoms administered in the beginning, middle and end of session. Subjects asked to rate strength of sensations on 4 point scale. Symptoms assessed were dizziness, headache, fatigue, tingling, redness, warmth	There were no significant differences between mean values for subjective ratings between exposure on and exposure off situtations.